



Enabling HIV Sexual Transmission with Antiviral Drug-Based Inhibitors

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ABOUT THE STUDY

The human immunodeficiency virus, or HIV, needs to penetrate a vulnerable cell to proliferate, along with all other viruses. Blocking its replication is a huge medical priority around 2-3 million people contract HIV each year. Inside the vaginal epithelia or through rifts in the mucosal lining, virus in semen or mucosal fluids encounters susceptible cells to enter, such as T lymphocytes and dendritic cells. Once the virus has entered a cell, it can begin replication and produce offspring virus. If replication spreads from local lymphoid tissue to regional lymph nodes, then to gut-associated lymphoid tissue and blood, the host will become infected systemically [1].

Antiretroviral medications are mostly responsible for it too. These medications function in HIV patients by preventing the virus from entering specific cells in the body and replicating. Because they function against retroviruses like HIV, these medications are known as antiretroviral [2]. Protease inhibitors are one type of antiretroviral medication used to treat HIV. These medications are designed to lower the amount of HIV virus in the body known as the viral load to undetectable levels. This reduces HIV progression and aids in the treatment of symptoms. HIV's principal goal is to replicate itself as many times as possible [3]. HIV, on the other hand, obviously lacks the machinery to replicate. However, it initiates its genetic material inside CD4 cells, which are immunological cells in the body. These cells are then used as an HIV viral factory. Protease is an enzyme that helps the body replicate HIV. Protease inhibitors stop protease enzymes from working. As a result, protease enzymes are unable to play their function in permitting HIV to proliferate, disrupting the HIV life cycle. This can prevent the virus from spreading [4].

The essential position of HIV protease in viral growth and development makes it a very attractive target for drug design. The discovery of new and superior inhibitors has been considerably aided by the enormous number of solved HIV protease protein structures. Saquinavir, indinavir, ritonavir, nelfinavir, amprenavir, fosamprenavir, lopinavir, atazanavir, tipranavir, and darunavir are among the HIV protease inhibitors

authorized by the FDA. Unfortunately, the majority of inhibitors have negative effects when used long-term [5].

HIV protease inhibitor-induced metabolic syndromes, such as dyslipidemia, insulin resistance, and lipodystrophy, lipoatrophy, as well as cardiovascular and cerebrovascular disorders, are the most prevalent side effects. Monotherapy with a protease inhibitor is linked to a little improvement in body fat distribution [6]. However, there were no significant between-group differences in adverse outcomes of antiretroviral therapies among HIV protease inhibitor standard treatment and combinations of protease inhibitors with HIV integrase inhibitors or Nucleoside Reverse Transcriptase Inhibitors (NRTIs), indicating that HIV protease inhibitors may be responsible for the most serious adverse effects [7].

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